Fogarty catheter and ice saline lavage may also be useful in patients with persistent bleeding.[1]

Fortunately, most episodes of hemoptysis can be controlled with conservative management, and definitive therapy can be delayed.[18] Whereas some think that surgical resection is still the treatment of choice for most if not all anatomically circumscribed lesions,[1,2] embolization therapy using either detachable balloons[7] or steel coils[19] has been advocated by others. Embolization therapy is obviously a less invasive procedure than thoracotomy and has a high success rate in terminating active hemoptysis. The potential for recurrence, however, remains controversial. Embolization has been successfully performed during pregnancy[11] and should be strongly considered at the time of angiography.

In conclusion, pulmonary arteriovenous malformations should be considered in the differential diagnosis of hemoptysis during pregnancy. Two-dimensional echocardiography with bubble contrast is a sensitive means of detecting the disorder and is without any known risk to a pregnant woman or fetus. Pulmonary angiography can be used to confirm the findings, and embolization can then be used as definitive treatment either immediately in life-threatening cases of hemoptysis or following delivery if the patient becomes stable with conservative treatment.

REFERENCES

1. Thompson AB, Teschler H, Rennard S: Pathogenesis, evaluation, and therapy for massive hemoptysis. Clin Chest Med 1992; 13:69-82

2. Knott-Craig CJ, Oostuizen JG, Rossouw G, Joubert JR, Barnard PM: Management and prognosis of massive hemoptysis. J Thorac Cardiovasc Surg 1993; 105:394-397

3. DePace NL, Betesh JS, Kotler MN: 'Postmortem' cesarean section with recovery of both mother and offspring. JAMA 1982; 248:971-973

4. Burke CM, Safai C, Nelson DP, Raffin TA: Pulmonary arteriovenous malformations: A critical update. Am Rev Respir Dis 1986; 134:334-339

5. White RI: Pulmonary arteriovenous malformations: How do we diagnose them, and why is it important to do so? Radiology 1992; 182:633-635

6. Dines DE, Arms RA, Bernatz PE, Gomes MR: Pulmonary arteriovenous fistulas. Mayo Clin Proc 1974; 49:460-465

7. White RI Jr, Lynch-Nyhan A, Terry P, et al: Pulmonary arteriovenous malformations: Techniques and long-term outcome of embolotherapy. Radiology 1988; 169:663-669

8. Metcalfe J, McAnulty JH, Ueland K: Cardiovascular physiology. Clin Obstet Gynecol 1981; 24:693-710

9. Elliott JA, Rankin RN, Inwood MJ, Milne JK: An arteriovenous malformation in pregnancy: A case report and review of the literature. Am J Obstet Gynecol 1985; 152:85-88

10. Laroche CM, Wells F, Shneerson J: Massive hemothorax due to enlarging arteriovenous fistula in pregnancy. Chest 1992; 101:1452-1454

11. Gammon RB, Miksa AK, Keller FS: Osler-Weber-Rendu disease and pulmonary arteriovenous fistulas. Chest 1990; 98:1522-1524

12. Moore BP: Pulmonary arterio-venous fistula. Thorax 1969; 24:381

13. Swinburne AJ, Fedullo AJ, Gangemi R, Mijangos JA: Hereditary telangiectasia and multiple pulmonary arteriovenous fistulas—Clinical deterioration during pregnancy. Chest 1986; 89:459-460

14. Dines DE, Seward JB, Bernatz PE: Pulmonary arteriovenous fistulas. Mayo Clin Proc 1983; 58:176-181

15. Dinsmore BJ, Gefter WB, Hatabu H, Kressel HY: Pulmonary arteriovenous malformations: Diagnosis by gradient-refocused MR imaging. J Comput Assist Tomogr 1990; 14:918-923

16. Barzilai B, Waggoner AD, Spessert C, Picus D, Goodenberger D: Two-dimensional contrast echocardiography in the detection and follow-up of congenital pulmonary arteriovenous malformations. Am J Cardiol 1991; 68:1507-1510

17. Milsom I, Forssman L: Factors influencing aortocaval compression in late pregnancy. Am J Obstet Gynecol 1984; 148:764-771

18. Jones DK, Davies RJ: Massive hemoptysis—Medical management will usually arrest the bleeding. BMJ 1990; 300:889-890

19. Hartnell GG, Jackson JE, Allison DJ: Coil embolization of pulmonary arteriovenous malformations. Cardiovasc Intervent Radiol 1990; 13:347-350

Electrocardiographic Changes Associated With Anaphylaxis in a Patient With Normal Coronary Arteries

MARY BETH ENGRAV, MD
MICHAEL ZIMMERMAN, MD
Portland, Oregon

CLINICIANS HAVE DOCUMENTED the occurrence and have postulated mechanisms for ischemic electrocardiographic (ECG) changes during anaphylactic reactions. Coronary angiography has shown that ECG changes do not invariably correspond to underlying coronary artery abnormalities. We report the case of an older woman who, during an initially undiagnosed anaphylactic reaction, had ECG changes suggesting acute myocardial ischemia. Angiography showed normal coronary artery anatomy.

Report of a Case

A 65-year-old woman was brought to the emergency department by paramedics after she suddenly collapsed while walking down the street. When paramedics arrived, she was diaphoretic and pale; her blood pressure was 80 mm of mercury systolic, her pulse rate was 80 beats per minute, and respirations were 20 per minute. En route to the hospital, her serum glucose level was 120 mg per dl (6.7 mmol per liter).

On arrival, the patient was nonresponsive, with no spontaneous eye opening. She remained diaphoretic and pale. Her temperature was 35°C, her blood pressure was 60 mm of mercury by palpation, and her pulse rate was 126 and regular. There was no evidence of trauma to the head. Both pupils were 4 mm and reactive to light. Respirations were shallow, and no wheezing, rales, or rhonchi were heard. Examination of her heart revealed no murmur or gallop. There was no edema or cyanosis of the extremities. Femoral and carotid pulses were palpable, although radial and dorsalis pedis pulses could not be felt. Her skin was without rash or lesions. Corneal and gag reflexes were present. The Babinski response was down-going bilaterally, and the patient withdrew all four extremities to pain. She would not answer questions and did not vocalize with painful stimuli. Her rectal tone was normal. Stool was negative for occult blood.

By phone, the patient's daughter and husband reported that the patient had appeared well when she left home earlier that day. They reported a history of hypercholesterolemia and hypothyroidism. Current medications included conjugated estrogens, medroxyprogesterone

(Engrav MB, Zimmerman M: Electrocardiographic changes associated with anaphylaxis in a patient with normal coronary arteries. West J Med 1994; 161:602-604)

From the Emergency Department, St Vincent Hospital and Medical Center, Portland, Oregon.

Reprint requests to Mary Beth Engrav, MD, Emergency Department, St Vincent Hospital and Medical Center, 9205 SW Barnes Rd, Portland, OR 97225.

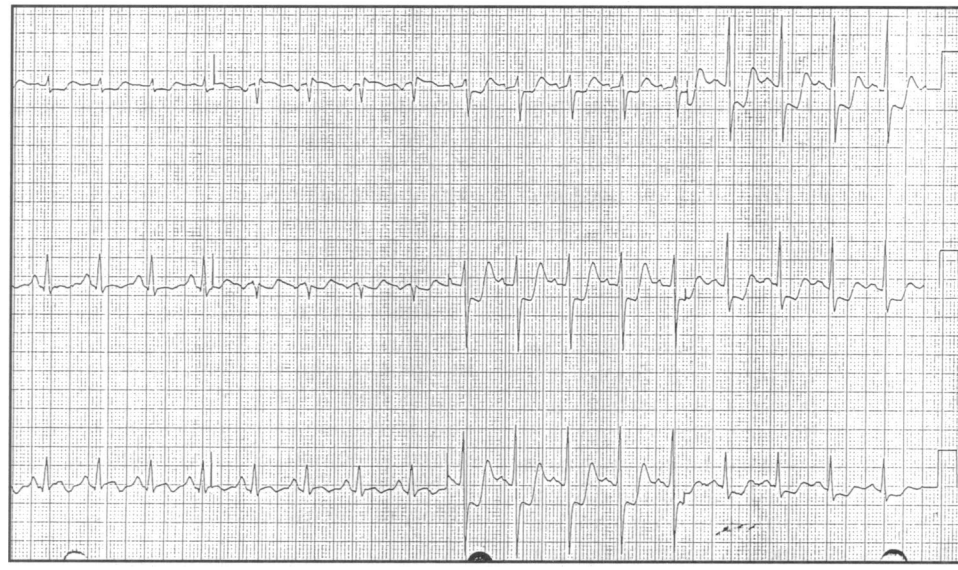

Figure 1.—The initial electrocardiogram suggests ischemia.

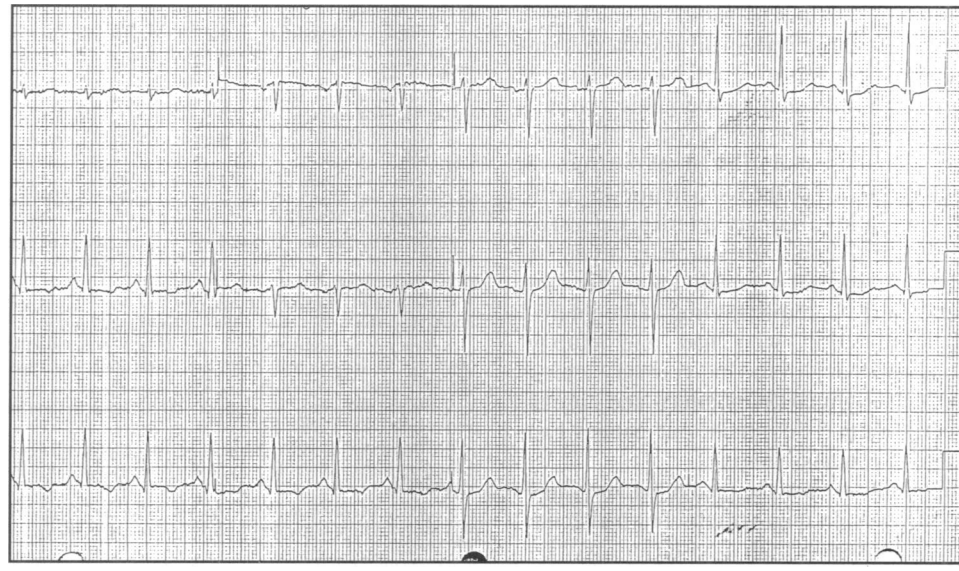

Figure 2.—A second electrocardiogram shows that the ST segments have returned to normal.

acetate, and levothyroxine sodium. The family history was pertinent for coronary artery disease in her mother and sister.

On the cardiac monitor, the patient remained in sinus tachycardia without ectopy. Changes on the ECG were interpreted as showing an acute posterior myocardial infarction (Figure 1). The following laboratory values were elicited: serum sodium, 139 mmol per liter; potassium, 3.1 mmol per liter; chloride, 108 mmol per liter; bicarbonate, 24 mmol per liter; urea nitrogen, 8.2 mmol per liter (23 mg per dl); creatinine, 106 μmol per liter (1.2 mg per dl); and glucose, 8.2 mmol per liter (147 mg per dl). The leukocyte count was 12.4×10^9 per liter (12,400 per mm^3), the hemoglobin level was 116 grams per liter (11.6 grams per dl), the hematocrit was 0.345 (34.5%), and a platelet count was 234×10^9 per liter (234,000 per mm^3). The initial creatine kinase level was 189 IU per liter, with 0.055 (5.5%) MB fractions. A chest x-ray film was normal.

After the initial ECG, a cardiology consultation was obtained. The patient received boluses of saline solution in 250-ml increments to a total of 2 liters, and dobutamine hydrochloride was added. The systolic blood pressure did not exceed 70 mm of mercury systolic. The patient's mental state improved slightly. She provided short, irritable responses to questions, and she denied having pain or other symptoms. Coronary angiography showed normal coronary artery anatomy and a left ventricular end-diastolic pressure of 11 mm of mercury. No vasospasm was seen.

Shortly after the angiogram was completed, the patient recalled being stung by a bee as she was walking to

the library. She had no previous history of allergy to bee stings. Her blood pressure was improving without further intervention, and she was given methylprednisolone sodium succinate (Solu-Medrol) and diphenhydramine hydrochloride (Benadryl) intravenously. A second ECG showed a return to normal of previous changes (Figure 2), and serial creatine kinase and MB values remained normal. After discharge the patient was referred to an allergist for Hymenoptera skin testing.

Discussion

Electrocardiographic changes consistent with acute myocardial infarction have been noted with anaphylactic reactions.[1-7] We found three reports of cases in which coronary angiography was done after anaphylaxis occurred.[1,2,8] One was in a 64-year-old man who had no history of heart disease and in whom anaphylaxis developed after he was given cefoxitin.[2] His ECG revealed acute inferior-wall injury. Serial creatine kinase enzyme levels were within normal limits. His angiography showed patent coronary arteries, although he had an anomalous origin of his right coronary artery from the left sinus of Valsalva, which possibly predisposed him to an inferior-wall myocardial injury pattern. A second case was of a patient who had an anaphylactic reaction to iodinated radiographic contrast material and had an acute inferior-wall myocardial infarction.[8] Angiography demonstrated substantial obstructive disease of the diagonal branch of the left anterior descending and distal left circumflex coronary arteries and a saccular aneurysm of the proximal right coronary artery. A third case involved a 76-year-old man with idiopathic anaphylactic shock and an ECG demonstrating acute inferolateral myocardial infarction who had angiography showing no notable narrowing of the left coronary artery, moderate stenosis of the right coronary artery, and normal left ventricular wall motion.[1] As noted, our patient had normal coronary arteries on angiography.

Our case is important because the patient had no signs of coronary artery disease on angiography but had acute ST changes on ECG consistent with a posterior myocardial infarction. Different mechanisms have been proposed to explain such changes occurring during anaphylaxis. In the past it had been postulated that the epinephrine given during an acute anaphylactic reaction caused subsequent ECG changes.[9] In our patient, ECG changes were recorded before the patient received any epinephrine. Hypoxia had been suggested in the past as a cause of the ischemic ECG changes, although studies done in intubated, well-oxygenated animals show that myocardial ischemia occurs before the bronchoconstriction seen in anaphylaxis.[6,10,11] Another proposed mechanism is vasospasm. Anaphylactic reactions of isolated perfused guinea-pig hearts show coronary vasoconstriction.[12] Different mediators causing coronary vasospasm have been proposed, including histamine, platelet-activating factor, leukotrienes C_4, D_4, and E_4, and prostaglandin SRS-A.[5,13-15] Mast cells have been found in high concentrations in atheromatous plaques.[16] Vasospasm has not been documented in humans during coronary angiography and was not seen in this patient.

This case report documents ECG changes consistent with myocardial ischemia during an anaphylactic reaction in a patient who was found at coronary angiography to have normal coronary arteries. Our patient's creatine kinase levels remained normal, and her ECG changes resolved to normal, consistent with the findings in other patients with ECG changes related to anaphylaxis and no history of coronary artery disease.

REFERENCES

1. Wong S, Greenberger PA, Patterson R: Nearly fatal idiopathic anaphylactic reaction resulting in cardiovascular collapse and myocardial infarction. Chest 1990; 98:501-503

2. Austin SM, Barooah B, Kim CS: Reversible acute cardiac injury during cefoxitin-induced anaphylaxis in a patient with normal coronary arteries. Am J Med 1984; 77:729-732

3. Nagaratnam N, Husodo H, James WE: Electrocardiographic changes following bee-sting anaphylaxis. J R Soc Med 1988; 81:420-421

4. Assem ESK: Anaphylactic reactions affecting the human heart. Agents Actions 1989; 27:142-145

5. Felix SB, Baumann G, Hashemi T, et al: Effects of histamine H_1-receptor blockade on respiratory and cardiac manifestation of systemic anaphylaxis. Agents Actions 1991; 33:349-358

6. Felix SB, Baumann G, Niemczyk M, et al: Effects of histamine H_1- and H_2-receptor antagonists on cardiovascular function during systemic anaphylaxis in guinea pigs. Agents Actions 1991; 32:245-252

7. Antonelli D, Koltun B, Barzilay J: Transient ST segment elevation during anaphylactic shock. Am Heart J 1984; 108(pt1):1052-1054

8. Druck MN, Johnstone DE, Staniloff H, McLaughlin PR: Coronary artery spasm as a manifestation of anaphylactoid reaction to iodinated contrast material. Can Med Assoc J 1981; 125:1133-1135

9. Mehta D, Warwick GL, Goldberg MJ: QT prolongation after ampicillin prophylaxis. Br Heart J 1986; 55:308-310

10. Capurro N, Levi R: The heart as a target organ in systemic allergic reactions: Comparison of cardiac anaphylaxis in vivo and in vitro. Circ Res 1975; 36:520-528

11. Felix SB, Baumann G, Berdel WE: Systemic anaphylaxis—Separation of cardiac events from respiratory and peripheral events. Res Exp Med (Berl) 1990; 190:239-252

12. Levi R: Cardiac anaphylaxis: Models, mediators, mechanisms, and clinical considerations. Clin Immunol 1988; 1:93-105

13. Felix SB, Baumann G, Hashemi T, Niemczyk M, Ahmad Z, Berdel WE: Characterization of cardiovascular events mediated by platelet activating factor during systemic anaphylaxis. J Cardiovasc Pharmacol 1990; 15:987-997

14. Forman MB, Oates JA, Robertson D, Robertson RM, Roberts LJ II, Virmani R: Increased adventitial mast cells in a patient with coronary spasm. N Engl J Med 1985; 313:1138-1141

15. Burke JA, Levi R, Guo ZG, Corey EJ: Leukotrienes C_4, D_4, and E_4: Effects on human and guinea-pig cardiac preparations in vitro. J Pharmacol Exp Ther 1982; 221:235-241

16. Pomerance A: Periarterial mast cells in coronary atheroma and thrombosis. J Pathol Bacteriol 1981; 70:256-261